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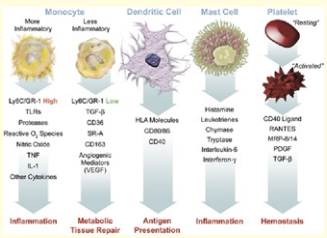
Inside This Issue

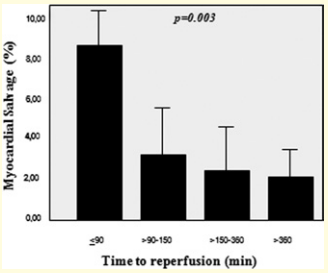
STATE-OF-THE-ART PAPER



VIEWPOINT

(continued on page A-30)

CLINICAL RESEARCH



INTERVENTIONAL CARDIOLOGY

CMR Study Demonstrates Benefits of Prompt Primary PCI 2145

Marco Francone, Chiara Bucciarelli-Ducci, Iacopo Carbone, Emanuele Canali, Raffaele Scardala, Francesca A. Calabrese, Gennaro Sardella, Massimo Mancone, Carlo Catalano, Francesco Fedele, Roberto Passariello, Jan Bogaert, Luciano Agati

Francone and colleagues investigated the relationship between myocardial damage and time to reperfusion using cardiovascular magnetic resonance (CMR) in patients with ST-segment elevation myocardial infarction who underwent primary percutaneous coronary intervention (PCI). Myocardial edema and necrosis were quantified as the area at risk and infarct size, respectively; salvaged myocardium was defined as the difference between the two. Shorter time to reperfusion (<90 min) was associated with smaller infarct size and larger salvaged myocardium. Salvaged myocardium markedly decreased when reperfusion occurred >90 min after symptom onset. Time to reperfusion determines the extent of reversible and irreversible myocardial injury, with 90 min from symptom onset being an important threshold.

Editorial Comment: Richard W. Smalling, p. 2154

INTERVENTIONAL CARDIOLOGY

One Dose of Atorvastatin Reduces Rates of Periprocedural MI 2157

Carlo Briguori, Gabriella Visconti, Amelia Focaccio, Bruno Golia, Alaide Chieffo, Alfredo Castelli, Marco Mussardo, Matteo Montorfano, Bruno Ricciardelli, Antonio Colombo

Some data suggests that statins can reduce the rates of periprocedural myocardial infarction (MI) in patients undergoing percutaneous coronary intervention (PCI). Briguori and colleagues hypothesized that this benefit is due to their "pleiotropic" effects, independent of cholesterol reduction, and that this benefit could develop within 24 h. Statin-naive subjects undergoing elective PCI were randomized to either a single 80-mg dose of atorvastatin, given within 24 h of the procedure, or usual care. Periprocedural MI, defined as a creatine kinase-myocardial isoenzyme elevation >3 times the upper limit of normal, occurred in 9.5% of the atorvastatin group and 15.8% of the control group. Similar reductions were found with troponin elevation. A single dose of atorvastatin reduces the incidence of periprocedural MI in elective PCI.

Editorial Comment: Sotirios Tsimikas, p. 2164

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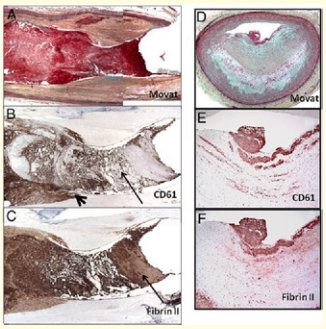
CORONARY ARTERY DISEASE

Autopsy Study: Microemboli and Microvascular Obstruction After Acute Coronary Thrombosis 2167

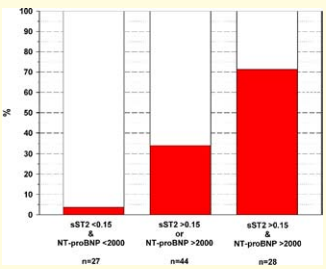


Robert S. Schwartz, Allen Burke, Andrew Farb, David Kaye, John R. Lesser, Timothy D. Henry, Renu Virmani

Schwartz and colleagues studied the histopathology of epicardial coronary artery plaque in patients dying from coronary artery occlusion and its relationship to microemboli and microvascular obstruction. Hearts from patients experiencing sudden coronary death underwent a histopathologic examination. There were 26 plaque ruptures and 21 plaque erosions in these 44 hearts, with a mean of 4.5 microemboli per heart. Vascular microemboli and microvascular obstruction occurred more often from plaque showing erosion rather than rupture, but intramyocardial microemboli were more common after plaque rupture. Microemboli and microvascular obstruction are common in patients dying of acute coronary thrombosis caused by plaque erosion.

BIOMARKERS

Soluble ST2 Predicts Risk of SCD in Patients With Chronic Heart Failure 2174



Domingo A. Pascual-Figal, Jordi Ordoñez-Llanos, Pedro L. Tornel, Rafael Vázquez, Teresa Puig, Mariano Valdés, Juan Cinca, Antoni Bayes de Luna, MAntoni Bayes-Genis, on behalf of the MUSIC Investigators

Pascual-Figal and colleagues used a large registry of patients with heart failure to determine if the soluble form of ST2 (sST2), an interleukin-1 receptor family member, predicts the risk of sudden cardiac death (SCD). A nested case-control study was performed on 36 cases of SCD and 63 controls (matched for age, sex, and left ventricular ejection fraction) enrolled in a registry of ambulatory heart failure patients. Concentrations of sST2 were higher among those who died. Combining sST2 and N-terminal pro–B-type natriuretic peptide (NT-proBNP) improved the accuracy: only 4% of patients with low sST2 and low NT-proBNP experienced SCD. The rate was 34% when either marker was elevated and 71% when both markers were elevated. sST2 may be useful for predicting the risk of SCD, especially when combined with NT-proBNP.

(*continued on page A-38*)

CONGENITAL HEART DISEASE

Percutaneous Stenting Compared to the Blalock-Taussig Shunt **2180**

Giuseppe Santoro, Giovanbattista Capozzi, Giuseppe Caianiello, Maria Teresa Palladino, Chiara Marrone, Gabriella Farina, Maria Giovanna Russo, Raffaele Calabrò

This study sought to determine if arterial duct (AD) stenting or a modified Blalock-Taussig shunt (MBTS) was more successful at stimulating growth in the pulmonary arteries (PAs) in neonates with congenital heart disease with duct-dependent pulmonary circulation. Subjects who had undergone one of the procedures were compared. The PA growth was angiographically assessed using a variety of indexes, and the right-to-left PA diameter ratio was measured as a surrogate of uniform growth. After 10 months, both options had promoted a significant increase in PA size; however, the surgical shunt caused a worsening of the left-to-right PA diameter ratio due to growth of the PA contra-lateral to the shunt. Percutaneous AD stenting appears to be as effective as MBTS in promoting PA growth and may result in more balanced pulmonary vascular development.

PRE-CLINICAL RESEARCH

PRE-CLINICAL RESEARCH

ALDH2 Ameliorates Acute Cardiac Toxicity of Ethanol **2187**

Heng Ma, Ji Li, Feng Gao, Jun Ren

Ma and colleagues hypothesized that acetaldehyde, which is the first metabolic product of ethanol, may be the toxic factor that causes alcoholic cardiomyopathy. They tested this hypothesis by generating a strain of transgenic mice with cardiac overexpression of mitochondrial aldehyde dehydrogenase-2 (ALDH2), which breaks down acetaldehyde. Wild-type and ALDH2 mice were injected with ethanol (3 g/kg, intraperitoneally), and cardiac function was assessed 24 h later using both Langendroff and cardiomyocyte edge-detection systems. The deteriorations in myocardial and cardiomyocyte contractile function were alleviated by ALDH2. Furthermore, ethanol-induced myocardial apoptosis, protein damage, and mitochondrial membrane potential depolarization were lessened in ALDH2 mice. This study demonstrates that ALDH2 may be protective against acute ethanol-induced cardiac toxicity.

Editorial Comment: Joel S. Karliner, p. 2197